Need For Therapeutic Anticoagulation In The Setting Of Penetrating Traumatic Brain Injury

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Introduction to Acute Traumatic Brain Injury

Overview:

Penetrating traumatic brain injury (PTBI) can be a disastrous mechanism of injury and has significant implications for ongoing management if the patient survives the primary injury. After ensuring hemodynamic stability and achieving hemostasis, the patient is likely to have both cognitive and functional deficits. A 29 year old male presented after sustaining a self-inflicted gunshot wound to the head. He was initially hemodynamically unstable, had minimal neurologic status, and had neurosurgical bleeding which was difficult to control due to superior sagittal sinus disruption. The patient remained bedridden, and although he received appropriate DVT prophylaxis, he developed a pulmonary embolus. The patient received therapeutic heparin, but as a result he developed intracranial hemorrhages necessitating multiple neurosurgical drain placements which were functionally short-lived. Due to these hemorrhagic episodes, his heparin was intermittently held and resumed, culminating in multiple hemorrhages, herniation, and death.

HPI:

A previously healthy 29 year old male presented via aeromedical transport after sustaining a reported self-inflicted gunshot wound.

Hospital course:

- HD 0: Emergent decompressive craniectomy, developed hemorrhagic hypotension from sagittal sinus. Surgery aborted, temporarily closed.
- HD 2: GCS improved from 3T to 10T, successfully extubated.
- HD 4: Coil embolization of the sagittal sinus performed.
- HD 5: Found to have occlusive left femoral DVT; IVC filter placed.
- HD 6: Returned to OR for removal of packing and definitive closure.
- HD 8: Started chemical DVT prophylaxis.
- HD 12: Found to have PE in superior trunk of the right pulmonary artery.
- HD 13: Cleared by neurosurgery to start therapeutic heparin treatment.
- HD 15: Repeat CT head with evolving parenchymal hemorrhages. No neurological exam changes, GCS 13. Heparin drip discontinued.
- HD 16: Repeat imaging showed evolving hydrocephalus. External ventricular drain (EVD) placed.
- HD 18: Restarted heparin drip. EVD drained frank blood 3 hours later. Exam unchanged, heparin discontinued, STAT imaging obtained.
- HD 20: EVD stopped functioning. Replaced after PTT normalized. Heparin restarted after neurosurgical clearance.
- HD 22: EVD with frank blood, heparin held. No physical exam changes.
- HD 23: New bilateral EVD's placed, freely drained blood-tinged fluid. Postoperative CT showed new and increasing biventricular bleeding.
- HD 25: Heparin drip restarted, EVD's later stopped draining.
- HD 28: Acute decline in neurologic status, GCS 8, imaging with evidence of herniation. Reintubated for impending respiratory failure. Lumbar drain placed by Interventional Radiology. Overnight developed pulseless ventricular tachycardia, coded for 20 minutes before death pronounced.

ICU Management

Perform primary and secondary surveys; secure airway and restore hemodynamic stability if not already accomplished.

Assess surgical candidacy based on neurologic status and radiologic evidence of hemorrhage, hematoma, and any resultant mass effect producing midline shift or herniation. Epidural hematomas >30ml in volume, and subdural hematomas >10mm in thickness or with >5mm midline shift warrant neurosurgical intervention.[1]

Penetrating mechanisms of injury and other open skull fractures also dictate the need for debridement and dural closure.[1] Elevated ICP refractory to medical management can be remedied by decompressive craniectomy or placement of intracranial or lumbar drains.

Placement of invasive arterial or central venous catheters are placed after initial stabilization to tailor appropriate therapies. They are important adjuncts to monitoring and vascular access for the anesthesia team in the perioperative setting and are invaluable if the patient acutely decompensates.

Unfavorable Prognostic Indicators in PTBI[2]

- Older age
- **Suicide attempt**
- **Mode of injury** (**penetrating** vs tangential)
- Hypotension
- **Coagulopathy**
- Respiratory distress
- **Low GCS**
- Pupillary size and light reflex
- **Elevated ICP**
- **Imaging findings**

Concerning Clinical Findings

- Cushing's triad
  - Systolic hypertension
  - Bradycardia
  - Irregular respiration
- Unreactive pupils
- Anisocoria
- Decorticate, decerebrate posturing

Treatment Guidelines[3]

- Avoid hypotension (SBP < 90mmHg)
- Avoid hypoxemia (SaO2 ≤ 90%)
- Employ timely hyperosmolar therapy
- Maintain euthermia
- Avoid prolonged systemic antibiotic prophylaxis
- Start chemical DVT prophylaxis after multidisciplinary discussion
- Monitor ICP in survivable injuries
- Initiate treatment for ICP > 20mmHg
- Target cerebral perfusion pressures between 60-70mmHg
- Provide analgesics and sedatives during mechanical ventilation
- Initiate feeding by post-injury day 5-7
- Use anticonvulsants to decrease the incidence of seizures in the first week
- Reserve hyperventilation as a temporizing measure for elevated ICP
- Avoid routine use of steroids

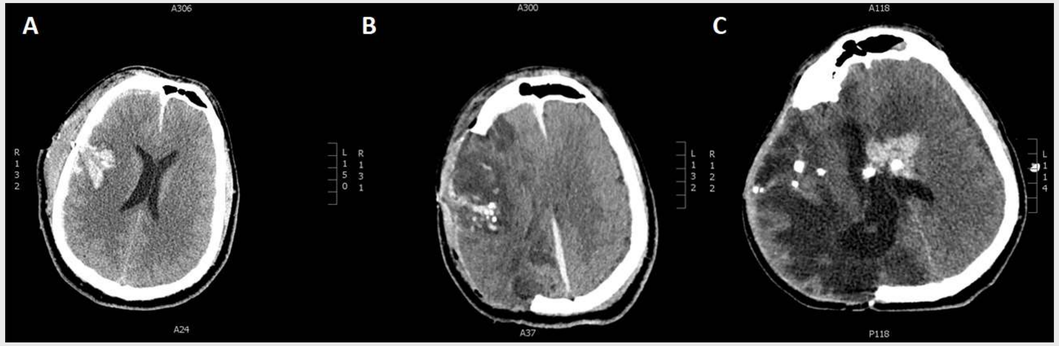

Figure 1: Non-contrasted computed tomography, axial views of the head and brain. A. Hospital Day 0; penetrating traumatic injury showing subarachnoid and intraparenchymal hemorrhages, and subdural hematoma with 6.8mm midline shift. B. Hospital Day 6; status-post craniectomy, with ischemic changes and retained bullet fragments. C. Hospital Day 28; status-post bilateral ventriculosotomy, evolving hemorrhage and ventriculomegaly demonstrated.

Discussion

The overarching goal in neurotrauma is to avoid secondary injury by means of maintaining physiologic stability and providing selective prophylactic treatment. Managing electrolytes, acid-base status, glucose and nutrition, coagulopathy, and seizure and VTE prophylaxis are essential to initial resuscitation and ongoing treatment.

Crystalloids are preferred over colloids to achieve euvolemia, as has been shown in the SAFE clinical trial.[4] Strict fluid and electrolyte management minimizes osmolar shifting and cellular edema. This patient did not immediately undergo definitive craniectomy, which underscores the importance of maintaining normal ICP to mitigate cerebral edema and herniation in the setting of a fixed-space calvarium.

Adhering to systolic blood pressure goals of 100-110 mmHg and cerebral perfusion pressures of 60-70 mmHg ensures end-organ perfusion while also limiting the evolution of intracranial hemorrhage.[3] Vasoactive medications and mechanical hyperventilation are commonly employed to artificially preserve these ranges by manipulating cerebral blood flow, especially in the setting of impending herniation.

Balancing VTE prophylaxis with the risk of evolving hemorrhage is challenging to accomplish, as patients with PTBI are often coagulopathic due to their primary injury.[5] Cerebral tissue contains high concentrations of procoagulant elements,[6] which may prevent the clinician from delivering chemical prophylaxis in the initial stages of recovery. Physical prophylaxis like IVC filters and pneumatic compression stockings offer some benefit but are not perfect measures. In the case of a large proximal pulmonary artery embolus, a thorough and candid discussion with the patient, family, and neurosurgical and ICU teams must occur prior to systemic treatment, because complications are indeed lethal.

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